PNEUMONIA OF DELAYED RESOLUTION AND FIBROID PHTHISIS IN CHILDHOOD.

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In Fagge's *Principles and Practice of Medicine* occurs the following passage:

"Addison[1] described certain rare and exceptional cases in which, after an illness of a few weeks, with all the evidences of consolidation present, the patient dies and the lung is found uniformly albuminized. 'The section is not soft, lacerable, and granular as in acute gray hepatization, but smooth, solid, and tough. The alveoli are full of fibrillated lymph, and the exudation cells may have begun to be granular and fatty. It is a condition of the lung very liable to break down, and thus constitute a case of rapid phthisis; and, on the other hand, it is a condition from which recovery may take place and the lung be completely restored.' Wilkes and Moxon,[2] after quoting this account, continue: 'That there is a chronic pneumonia of such a kind can scarcely be denied when it is remembered for how long a time all the signs of consolidation may endure, and then a complete restoration take place. We must, therefore, believe that there is a true chronic pneumonia whose origin is an ordinary inflammation and exudation into the alveoli, and whose appearance is best noted by the term uniform albuminous induration.' They add, however, that such chronic pneumonia may be of the lobular kind, and Addison himself said that this condition may be limited to one or a few lobules only; so that it may, perhaps, be doubted whether this 'least frequent of the permanent pneumonic indurations of the lung' is not, after all, an unusually extensive catarrhal pneumonia."

This communication is to illustrate the fact that similar retarded resolution may be met in childhood, and to narrate cases which are examples of this condition. Further, I shall depict the cirrhotic lung of simple inflammatory type, and its mode of formation from a preceding pneumonia.

According to Fagge, Dürr has recorded, in a volume published by Jürgensen at Tübingen, two cases which occurred in very young children, but in each of which the primary attack appeared to be one of lobar, not catarrhal, pneumonia. *Apropos* of this he says: "Probably, however, the pneumonia which leads to cirrhosis, instead of being lobar is as a rule lobular and catarrhal, secondary to measles or whooping-cough."

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[1] Guy's Hospital Reports, 1843, and in the volume of his collected papers published by the New Sydenham Society, p. 27.

[2] Pathological Anatomy, p. 338.

Lobar broncho-pneumonia, however, is very frequent in childhood; moreover, I have already pointed out that the histological difference between croupous and broncho-pneumonia[1] is merely one of degree, that it is not by any means well marked or striking, and that no difference of kind exists in the sense that in one case we have to do exclusively with a croupous inflammation, and in the other with a catarrhal. Nor can such an origin be denied on the ground, held by some, that in catarrhal pneumonia alone the tubes and areolar tissue are invaded with leucocytes. Such an invasion does occur in croupous pneumonia, and the inflammatory condition in children is not by any means necessarily limited to the alveoli.

In pneumonia, when the temperature has fallen to normal, the usual course is for the lung mischief to clear up. Clearance mostly takes about a week, occasionally a little longer, sometimes a day or two, but it may be more prolonged or apparently remain in abeyance.

Resolution is attended by reflux crepitation of moderate-sized râles, more or less marked, which may be detected on the day of the crisis. Sometimes fine crepitations are heard. Again, crepitations may never be present, the consolidation disappearing noiselessly. This, omitting pleuritic effusions, is the usual course of events; but resolution may be so retarded as to raise a suspicion of tuberculosis.

In the first place, it is necessary to realize the great frequency of apical pneumonia in childhood, or a pneumonia invading the root and reaching to the upper lobe and the apex of the lower lobe, and further, to be fully alive to the fact that resolution occasionally occurs tardily, and that sometimes the consolidated mass remains in a state of inertia for days, weeks, or even months. Such a sequence is not sufficiently recognized, and it is in such cases as these that the suspicion previously mentioned may crop up, and a hasty and unfavorable prognosis hazarded. It is to just such cases—like those I am about to relate—that I wish to draw attention, and to impress the desirability of exercising caution before condemning the lung—and, with the lung, the patient.

Before proceeding further, the following clinical illustration may be read with interest:

CASE I.—Charles R., aged two years and nine months, was found to have a consolidated left apex on September 20. His temperature was 98° F. He had suffered from cough for a month, and the last week had been much worse.

On the 22d instant it was discovered to be clearing a little. On October 8th there was still consolidation of the lung, with dulness and tubular breathing. The child was much better in general health. On the 11th the lung condition was much improved. Dulness had disappeared, though the note was not good. There was no tubular breathing. On November 4th he was discharged quite well.

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[1] "Croupous and Broncho-pneumonia in Children: their Histological Identity." By George Carpenter and W. H. Syers, M A., M.D. Cantab., M.R.C.P. Practitioner, vol. xlvi., No. 6.

Case II.—William S., aged twelve months, attended on April 28th with consolidation of the right, upper, and middle lobes. His temperature was 98.4° F. He had suffered from a cough, and had been wasting for five weeks. The child was rickety, and presented an enlargement of the liver and spleen.

In spite of the extreme consolidation, he did not look at all ill. On May 5th there was no change in the physical signs. Scott's ointment was applied over the consolidated area.

On June 2d he was stated to be "getting on beautifully," but that he suffered from a most troublesome cough. He did not make any further attendance.

Case III.—Elizabeth W., aged four years, was seen on January 12th, and found to have a consolidated right apex. Fourteen days previously had taken cold. Her temperature was 101.8° F., and ran up and down until the 28th, when the oscillations became less, up to the 31st, and from that date the temperature varied between 98° F. and 99° F. for the most part, always falling short of 100° F., until she was lost sight of, on March 21st.

On January 27th there was nowhere absolute dulness, but abundant coarse crepitations all over the right chest. Bronchial breathing was heard in the axilla. By February 4th the râles were not so abundant, but otherwise the physical signs were as before. On February 12th she was found to be gaining weight. On the 19th she was taking her food well and making flesh. Her right chest was nearly as resonant as the left. Deficient inspiration and bronchial expiration were detected, accompanied by a few râles only, and those chiefly in front.

She was much better on the 25th and still gaining flesh. There was no actual dulness, but deficient resonance with distant bronchial breathing over the right scapular spine and above the right nipple. Fine, moist râles were detected over the right base up to the angle of the scapula. March 27th she was sent to St. Agatha's Home, Beckenham.

Case IV.—Annie G., aged three years, was seen on December 16th. There was a family history of rheumatism and phthisis. Her illness commenced six weeks previously, and she had been gradually getting worse. There were tubular breathing, dulness, and bronchophony of the left apex. The temperature was then a trifle raised, but fell on the 19th, and remained down all through except during a slight attack of bronchitis and also one of tonsillitis. On December 28th, the resonance, although still diminished very decidedly, was more than before. It was tympanitic where previously the dulness was nearly absolute. Posteriorly, there was very fair respiration in the supraspinous fossa. A note on March 13th recorded that she had gained weight since her admission, but the physical signs in the chest, although varying somewhat from time to time, had not materially changed. There was then almost absolute dulness beneath the left clavicle, and hardly anything to be heard. Posteriorly, there was more resonance than formerly, but very little breathing was detected. The bronchial breathing was much less than when first seen. The question of a localized collection of fluid suggested itself, but she seemed to have so little the matter with her that it was not thought worth while to explore. She was discharged on March 16th. Her mother, who subsequently attended as an out-patient, informed me that the child remained perfectly well until she caught measles sixteen months later. She had a trifling cough, but never brought up phlegm or blood. Her appetite was excellent, and she was in good condition. Toward the end the child had diarrhœa, some ten or twelve motions a day, and convulsions which lasted two days, and she died some three weeks after the onset of measles.

Case V.—Elizabeth S., aged six months, a congenital syphilitic, was brought on March 31st. Consolidation of the right upper, middle, and part of the lower lobe was found.

On April 3d, reported to have vomited "a deal of phlegm" the day previous. Her temperature was 99.8° F. Tubular breathing was now present at the extreme base. On April 17th she was "a deal better." On May 1st nettlerash made its appearance. As the physical signs remained unaltered, iodide of potassium ointment was ordered for the chest. By June 5th the chest was unaltered and the "cough was very bad." In spite of this, the infant was perfectly lively and bright. On June 30th there was still dulness over the right apex in front and behind, but not extending so far down as before. Tubular breathing and consonating râles were heard over the dull area. Dulness, tubular breathing, and consonating râles were detected over the upper lobe on August 18th. September 8th, the temperature was 101° F., the cause not being apparent. She was gaining flesh. I then took her into the hospital for closer observation, and she was an inmate until the 14th. Her temperature remained subnormal during the whole of this period. On September 18th she was obviously rickety; she looked well enough, however, and was quite bright and lively. The spleen was enlarged a trifle. On October 9th she was much better, but still suffered from urticaria. Her temperature was subnormal, and as far as the mother could judge, she was "in perfect health and getting fatter." The chest condition remained *in statu quo.* An ophthalmoscopic examination gave negative evidence. On November 24 I found a note by my former clinical assistant, Dr. W. H. Millar, to the effect that the right apex was a trifle dull and some crepitations were detected over it. All over the right lung the air entered badly in comparison with the left, and the breath sounds were weak. Extra puerile breath sounds were detected on the left. She was not brought again.

Recently I wrote to the patient's mother and received the following reply: "Our baby died on the 1st of January with convulsions, through being accidentally scalded on the legs."

Case VI.—Jessie S., aged five years and six months, was brought with a consolidated left apex and a temperature of 101.2° F., September 26th. October 3d the temperature was found normal and the physical signs were as before. October 10th she was progressing favorably, had a better appetite, and was improved in every way. The physical signs remained unchanged. She did not attend again.

Case VII.—James D., aged one year and eleven months, was brought with empyema of the left side on March 5th. Four fluid ounces of pus were evacuated, and consolidation of the upper lobe was left after the operation. On May 31st there were still tubular breathing, dulness, bronchophony, and some râles over this area. On June 5th the tube was removed, and on the 19th the resonance was very fair indeed all over, the breath sounds were vesicular and all signs of consolidation had completely disappeared.

CASE VIII.—Ethel M., aged six years, was first seen February 16th. The left upper lobe was solid, the temperature 103.2° F. On February 23d, the temperature was 100° F., the physical signs being as before. On March 5th, temperature was 98.2 F., and there was still consolidation. Dulness and tubular breathing were present on March 10th, but she was bright and lively. On the 23d, the lung was still dull and the breath sounds were of cog-wheel rhythm; but by April 6th the breath sounds were quite free and clear in every respect.

CASE IX.—James C., aged one year and six months, was seen on August 5th. He was said to have been ailing since birth, with his chest. His present illness commenced a month previously with diarrhœa and a cough. On examination, the breathing was loudly tubular at the right apex, back and front, extending down to the angle of the scapula posteriorly. There was deficient expansion, and coarse crepitations were heard below the scapular angle. He died on the 18th.

Post-mortem. The right pleura was adherent in several places. The surface of the right lung, the upper and lower lobes especially, were rough from adhesions. All the lobes were hard and of a dark-red color. The upper sank in water, the lower floated. The left lung and pleura were normal. All the organs were healthy. No tubercles detectable anywhere.

CASE X.—John M.,[1] aged one year and eight months, attended with an empyema on the left side, on October 29th. Seven ounces of pus were evacuated, together with thick pieces of caseating material, the upper lobe being left with signs of consolidation.

On November 5th the upper lobe was still solid, giving dulness and tubular breathing. The tube was discarded on January 25th, and he was sent to a convalescent home on March 9th, cured as regards the empyema. He was again examined on June 23d. The upper lobe was then absolutely dull, with marked tubular breathing, bronchophony and increased cry-fremitus. The resonance was deficient elsewhere in comparison and the entry of air not so good. The breath sounds were vesicular.

On August 9th the chest was of fluid dulness; in the upper axillary region there was deficient entry of air, and distant tubular breathing. An exploratory puncture went into solid lung. Scott's ointment was then ordered to be rubbed into the chest over the affected area, for ten minutes, night and morning.

On August 27th there was a note to the effect that since the commencement of mercurial inunction a marked improvement in the chest signs had taken place. The chest area corresponding to the upper lobe was still deficient in resonance in comparison with the other side, but this resonance was mingled with dulness. Breath sounds were then vesicular over this area; there was fair entry of air, and a few râles to be heard. The remainder of this side of the chest was deficient in resonance in comparison with the other side, but the entry of air was good, though not quite so marked as on the healthy side, and the breath sounds were vesicular. He was sent to Brighton the following day. The students in my class will remember seeing this case in October last, when I narrated his history as well as I could then remember. His chest was perfect as regards physical examination, and, with the exception of an adherent scar, there was absolutely nothing left to tell the tale of his former trouble. Consolidation lasted then, for certain, during a period of ten months, and probably did not completely disappear for some weeks, perhaps longer, after that examination.

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[1] This case was mentioned in International Clinics, January 1893. Article, "Pleurisy in Childhood," by the author.

Both in this case and in Case VII. the quantity of pus was small—far too little to bring about collapse of the apices. Such are merely instances of purulent pleurisy in association with pneumonia—a not at all infrequent starting-point of that disease in childhood.

In *International Clinics* (for January, 1893), when dealing with "Pleurisy in Childhood," I have already expressed my views, in these terms: "Such might pass for apical consolidation, and so it is—usually fibrinous, sometimes due to compression, occasionally tuberculous," which have, unfortunately, owing to a printer's error, been translated into: "Such might pass for apical consolidation, and so it is, usually fibrinous, but occasionally this compresson of the lung by bands of lymph causes tuberculosis"—a statement which, of course, was never intended.

CASE XI.—James R., aged twelve months, was first seen on March 11th. He had suffered from cough for six or seven weeks, and had snuffled since birth. His mother had had two miscarriages. His brothers and sisters were syphilitic. About six weeks back he had a "bad chest, was hot, feverish, experienced difficulty in breathing, and suffered from thirst." Three weeks previous "lumps appeared on the hand and wrist." He had consolidation, tubular breathing, dulness, and bronchophony of the left upper lobe and apex of the lower. His rib ends were beaded and the anterior fontanelle one and a half inches long. The spleen was one finger below the costal margin, and his liver could just be felt. There were cutaneous gummata on the left knuckles, and a gumma above the left wrist on the radial aspect, also one above the right wrist, palmar aspect. Temperature 101.6° F., pulse 156, respirations 80. I took him into the hospital, and when there he was found to have green and offensive diarrhœa. On March 30th the following note appears: "The temperature now registers 99.2° F., the pulse is 154, and the respirations 44." The abscesses on the wrist were opened, as they were on the point of bursting. By April 2d he had rapidly lost flesh. The lung was then *in statu quo.* In the afternoon the breathing became labored, the pulse very feeble and irregular, and death supervened. The presence of cutaneous gummata at one time led to the suspicion that this might be an instance of syphilitic disease of the lung; but the complete absence of reaction to treatment, and the large size, considerably modified these ideas.

Post-mortem. There were thin adhesions of the upper, and upper half of the lower lobe on the left side—the lobes were united. The upper lobe and upper part of the lower lobe were of a red color and consolidated. The bronchial tubes were thickened, dilated, and contained pus. Radiating from the bronchial tubes were scars of different sizes passing in various directions, and intersecting the solid areas in varying degrees. The microscope showed a combination of croupous and catarrhal products. Here there were masses of unaltered fibrin; in other situations, fibrinous masses with catarrhal cells; in others, again, catarrhal cells alone. The bronchial tubes were thickened, and presented more or less catarrhal inflammation. The above lesions were situated for the most part around the bronchial tubes. Some areas were quite healthy. In the areas of consolidation were noted proliferation of the alveolar walls and commencing fibroid changes. On the whole the catarrhal products were in excess of the croupous.

Judging from the shape of some of the alveoli, collapse had occurred. The autopsy further proved that this was a case of phthisis—although not apparent before microscopical examination—occurring in a syphilitic child. The gummata, which behaved somewhat differently to syphilitic gummata, probably owed their origin to a tubercular process.

On more than one occasion I have seen similar lung implication in young children; but here again, although not very marked, naked-eye gray or yellow tubercles were seen sparsely scattered throughout the affected area, leaving no doubt as to the nature of the process.

And here I must pause to say a word or two on syphilitic pneumonia of infants. Cases have been recorded by Greenfield[1]—though in this case there is no positive proof of a syphilitic origin—one aged twelve months, and one by Mr. Charters J. Symonds,[2] aged one month. The former showed consolidation of the right lung of a yellowish-white color, with minute bands of fibrous tissue running through it. The latter where attacked was solid and fleshy. Both showed extreme fibronucleated growth and dilated thin-walled capillaries. Still remaining air vesicles were seen with and without epithelial proliferation. Although the child was an obvious congenital syphilitic, the microscope showed that the process was after all a tubercular one. At one time I thought it might be of syphilitic origin. Cases of this condition have also been described by Virchow, Wagner, Robin, and Wilkes and Moxon.

In my experience, cases of retarded resolution in association with pneumonia have been found, with a solitary exception of basic pneumonia, in those showing an apical, or the upper and part of the lower lobe distribution.

Given, then, such a case, its nature and the probable sequence of events have to be determined. It must be borne in mind that fluid in the shape of pus sometimes limits itself to the apex. If the case has been under observation since the commencement, then the gradual dampening of the signs of consolidation would lead to a suspicion of a fluid collection. Bronchophony and tubular breathing, previously well marked, become distant, or a dampened vesicular murmur may take its place; or râles, previously sharp, metallic, and close to the ear, appear now more or less removed. There may be rises of temperature, with nocturnal exacerbations, or this may be raised but little, if at all. The child may or may not appear ill. If such are met with at the first examination, the possibility of a localized empyema must be borne in mind. When in doubt, do not hesitate to use the exploring-needle—its passage into a solid lung or cavity will solve the difficulty should any exist. This being negatived, is the consolidation of a simple nature, or does it owe its origin to a tubercular parentage?

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[1] Trans. Path. Society of London, vol. xxvii. p. 43.

[2] Ibid., vol. xxxvii. p. 124.

Continued rises of temperature, night-sweating, wasting, gurgling râles, progressive invasion of the lung, and the attachment of the fellow-apex, are in favor of phthisis. But the opposite lung may show no signs, and the gurgling râles may be merely an evidence of dilated tubes. Sometimes the difficulties are very great, and only to be solved in the dead-house. But inasmuch as tubercular affections in young children are so often of the generalized rather than the localized variety, the presence of other tubercular evidence must be remembered, viz., abdominal symptoms,[1] brain disturbances, tubercles in the choroid,[2] tubercular cutaneous lesions, testicular implication,[3] joint affections, and the like.

The fact that the consolidation remains *in statu quo;* that the particular signs referable to that condition persist, without much if any alteration; that the temperature remains normal; that the child, apart from auscultatory evidence, keeps well, both in mind and body, with obvious improvement in the general health, as gauged by appearance, increase of weight, and the mental condition, are all in favor of a simple consolidation of retarded resolution, whose final disappearance may or may not be heralded by crepitations. Time is all that is necessary, and time will clear up the nature of the disease. But in such cases as these an insidious fibrosis may be in motion—a fibrosis which, in some instances may be slight and but little progressive, or may pass on into a wide and far-reaching cirrhosis of the lung, with obliteration of its alveoli and general and extensive shrinkage of its bulk. Such a condition may or may not be associated with dilated tubes. Occasionally one sees in the post-mortem room slight scarring of a lung otherwise healthy, and it is just this scarring which may be a remnant of the condition previously mentioned, the branding of the organ, as it were, with the history of its former troubles. Such cirrhoses may be of a simple inflammatory nature, and it is with these that I am now dealing, or they may owe their origin to a tubercular process.

Tubercular fibroses are seen in children, and in the majority of instances are easily recognized as such; but in others the microscope is required for their definition and detection, and as an illustration of this I have narrated the case of J. R. If a microscopical examination had not been made, this lung, as far as outward appearances went, might have been readily passed into the group of simple cases.

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[1] "Tubercular Peritonitis, its Natural History and Treatment," by James F. Goodhart, M.D., F.R.C.P., and the author.

[2] "Tuberculosis of the Choroid," by the author; Illustrated Medical News, December 7 and 14, 1889, plates; Graduation Thesis, plates; British Medical Journal, 1890, plates.

[3] "Affections of the Testicles in Hereditary Syphilis," by the author, Practitioner, 1892.

Weber has narrated the cases of three children in whom the disease took origin in a croupous pneumonia, for which complaint he originally treated them.

In adults, such an onset has been occasionally observed. In a case of Charcot's,[1] death occurred three and one-half months after the onset of the acute disease, and the pathological changes were marked. A case of Osler's[2] died a month after the onset of the chill. The lung was uniformly solid and grayish in color. Some of the fibrin plugs were undergoing fatty degeneration, others fibroid organization, with great thickening of the alveolar walls. It was so in a young child under my care.[3] Cases are on record by Mayne,[4] by Coupland and Goodhart,[5] and Greene[6] has met with two cases in which the intra-alveolar exudation products were undergoing fibroid development. Wilson Fox insisted that bronchial dilatation is favorable to an indurative pneumonic process. Bronchial dilatation and peri-bronchitis are frequently met with in bronchitis or broncho-pneumonia, but only as part and parcel of the disease—of the same morbid inflammatory action.

Bronchial dilatation *per se*, in my opinion, is no more capable of inducing lung fibrosis than inflammatory lung mischief without bronchial attachment is competent to cause dilated tubes. As an illustration of instances of cirrhosis of the lung, I quote the following cases, proceeding then to discuss the methods of detection of this malady, and other details which have not been already gathered in the course of their narration.

CASE XII.—Isabel H.,[7] aged five years, was admitted on September 9, 1892. Her family history was negative. She had always been well until August, 1891, when she had pertussis, followed by measles and pneumonia in November, 1891. In July, 1892, she had a bad cough, with yellow expectoration streaked with blood, and was treated for this as an out-patient. She now has night-sweats and has wasted.

On admission, there was dulness all over the front of the left chest and behind up to the spine of the scapula. The breath sounds were deficient all over the front of the chest and behind, from the base to the level of the scapular spine. Distant tubular breathing, with a few crepitations, were heard. Above the spine of the scapula the breathing was loud and tubular, with râles. Cardiac impulse (?) displaced upward internal to the nipple. This supposed upward displacement was probably uncovering of the organ. Temperature 99° F. Respirations 48. Pulse 120. The case was thought to be one of empyema by the resident medical officer. The physical signs were identical; but it will be noticed that the heart was not displaced, a most important guide. Soon after admission an exploratory puncture was made in the eighth space, in the posterior axillary line, and about ten minims of pus mixed with air withdrawn. The child coughed after the needle was inserted, blood began to pour from her mouth and nostrils, and she died asphyxated.

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[1] Thèse de Paris.

[2] Principles and Practice of Medicine, 1892.

[3] "Croupous and Broncho-Pneumonia in Children, their Histological Identity." Practitioner, vol. xlvi., No. 6, p. 433.

[4] Dublin Hospital Gazette, May, 1857.

[5] Trans. Path. Soc. London, vol. xxv. p. 33.

[6] Pathology and Morbid Anatomy, 6th ed., p. 423.

[7] International Clinics, January, 1893, "Pleurisy in Childhood," by the author.

Post-mortem. The pericardium was hardly covered by the left lung at all.

Larynx: Full of frothy blood-stained mucus.

Bronchi: Full of blood-stained mucus, which extended on the right side as far as the medium-sized bronchi, and on the left into the smallest.

Right lung: Normal. Recent adhesions to thoracic pleura.

Stomach: Full of blood; a little blood in the duodenum. No sign of tubercle anywhere.

Left lung: This was small, showing a combination of cirrhosis, consolidation and induration of the lung tissue. The bronchial tubes were dilated and thickened, with scars radiating from them in all directions, and passing into cirrhotic, consolidated or indurated areas. Microscopically, these tubes showed an absence of mucous membrane. The basement membrane was infiltrated with leucocytes, and peri-bronchial infiltration was marked. Peri-bronchial, peri-vascular, and septal thickening were marked, the number of nuclei seen being variable. By these far-reaching fibroses the lung tissue proper was greatly curtailed. The bronchial walls, thickened and irregular in outline, look as if made up of granulation tissue. The changes in the alveolar walls themselves are various, from (though rarely) an absence of alteration to a most intense fibro-nuclear or fibroid induration, in which latter case whole areas are destroyed. The new tissue is exceedingly vascular. These changes appear to spread centrifugally from a small vessel or a bronchiole. The pleura is vascular and thickened, though but little. The alveoli show occasional very large granular cells with single nuclei. More frequently are seen catarrhal cells, or cells approaching leucocytes in appearance, or a combination of these elements. When the alveoli are much pressed up by fibrosis the catarrhal contents appear like a cubical epithelial lining. Further, some show an intra-alveolar material of varying size, which may or may not be stained fibrin masses with nuclei in them. Some show rifts, and others a certain fibrin-like network. The catarrhal cells do not seem to lend themselves to the new growth. They are there seemingly in a passive rather than an active condition, as far as fibrosis is concerned; nor can any organization of the fibrin be detected. Moreover, in some of the specimens they are very few and far between.

External to a large bronchial tube appears a tiny round-celled neoplasm, which seems to be undergoing fatty degeneration in the centre.

CASE XIII.—Catherine W., aged eight years, was kindly sent me by my friend, Dr. Hector Mackenzie, in December, 1892. Three years ago she suffered from whooping-cough. Her present illness "dated nineteen months back, with a hacking cough, worse in the morning." In February, 1892, she caught a fresh cold, and was kept in bed for two or three weeks, when she was transferred to the Brompton Hospital, where she remained three and a half months. At this time her cough became worse; but during her stay in hospital she derived considerable benefit.

Her father died of "inflammation of the lungs and pleurisy," and was ill one month. Two paternal uncles are said to have died from consumption.

Her mother expressed surprise at the child "seeming so well," and considered the "only trouble was the cough." She sweats a little at night occasionally, and expectorates about a quarter of a teacupful of yellowish offensive material three times a day, and generally after meals, after which she seems better. The matter is faintly streaked with blood. When she lies down at night she "feels a sensation of sickness," and then without any great amount of coughing or straining she brings up about one ounce of thickish, curdy pus. At odd intervals during the day she is troubled with a slight hacking cough.

Examination. The chest on the left side is considerably shrunken, and moves but little. The *cardiac impulse* is heaving in the anterior axillary line, in the sixth and seventh interspace. Over this impulse the sounds are normal. Above the nipple the first sound has a cantering rhythm, and there is a doubtful bruit in it of presystolic and systolic time. Attending this is a presystolic and systolic thrill. Over the infraspinatus muscle the heart sounds are loud and ringing.

The left side of the chest is uniformly dull from apex to base, and this dulness extends to within a finger's breadth of the left of the sternum. In front there are coarse bubbling râles, with coarse respiratory murmur, and behind, loud, tubular breathing from the apex to the eighth rib, as far forward as the posterior axillary fold. Below, the respiratory murmur is deficient, and the râles are smaller. On the right side the breath sounds are extra-puerile. The lung is hypertrophied, and extends well to the left of the left margin of the sternum. The sputum was examined for tubercle bacilli, but none were detected. The organs were normal, and there was no clubbing of the fingers and toes.

CASE XIV.—Amos D., aged seven years, was said to have had no illnesses up to July, but then he "caught a cold," at a school treat, which was followed by pneumonia, and ever since he had been ailing. In the middle of January following he came under my care. There was no history of syphilis, nor was there any phthisis in the family.

On admission he was found to be an emaciated, feeble child, with clubbed fingers. Respirations 50 to the minute, pulse 125, and temperature 99.6° F.

The cardiac impulse was in the fifth interspace diffused, but most marked in the nipple line. There was also an epigastric impulse. The area of dulness was well to the right of the sternum. The left chest gave tympanitic resonance in front above the third rib, and the breath sounds were vesicular. Below this dampened tubular breathing and weakened vesicular murmur were detected. Behind, in the supraspinous fossa were vesicular breath sounds; below, over the middle third or more, there was tubular breathing of an echoing quality, and below this again the breath sounds were dampened. The whole chest behind was dull. Over the opposite side the breath sounds were extra-puerile. The left chest was considerably shrunken, and there was marked compensatory lateral curvature. The urine was free from albumin. He suffered from troublesome cough, but there was but little expectoration. No tubercle bacilli. During the time he was under observation the physical signs changed from time to time. Thus, on one occasion, in the lower half, behind, and all over the front, with absence of cardiac dulness there was tympanitic resonance (stomach), and even whilst under examination the lung again became dull. Sometimes consonating râles were to be heard. On admission, the resident medical officer, suspecting pus, made an exploratory puncture in the ninth interspace, withdrawing a syringeful; but this was evidently from tapping a dilated tube.

Cirrhosis of the lungs is not so very uncommon in childhood. The chief signs and symptoms have been gathered by a study of the cases I have narrated. Retraction of the chest and compensatory lateral curvature may be but trifling. Thus, enormous dilated tubes may take up the portions of lung tissue, or the void left by a contracted lung relic may be filled up by an amyloid liver. Nor are dilated tubes invariably met with in cirrhotic lungs. Any portion of a lung may be attacked —the apex, the base, or both; usually, perhaps, the base when a single lobe is invaded.

In the case of implication of a single lobe there is no retraction of the chest, and the physical signs are those of consolidation, with consonating râles if there be any pus in the tubes, or gurgling râles if they be considerably dilated. There may be, with dulness, merely deficient entry of air without râles, or a few bronchitic signs.

Tubular or hollow breathing will depend upon the laryngeal intonation, and the latter may or may not denote dilated tubes. With one mode of respiration, the physical signs are those of fluid, with another the tubular character becomes apparent. The position of the heart is important. In left-sided lesions the organ may be uncovered, exposing a large portion, or the whole of its surface, with consequent diffusion of the impulse; or contracting adhesions with the lung, the heart may be displaced. Thus, if the right lung is attacked, the heart may be seen beating at the right nipple; if the left, pulsation may be detected up toward the left clavicle. Possibly anomalous murmurs from anatomical displacements, or a thrill, mayhap, may make their appearance. As time goes on, and as the fellow hypertrophied lung fails, the right side of the heart dilates, the fingers club, and the changes in the organs met with in this condition then make their appearance and stamp the case as one of secondary heart disease.

Prior to this, however, from the continual suppuration going on in the tubes, lardaceous changes in the viscera may occur, and the case become one of lardaceous disease. *Per se*, this condition of the lung gives rise, to commence with, to no particular symptoms, but the malady is detected by physical examination. The child may be fat rather than the reverse, and apart from shortness of breath on exertion there may not be apparently much the matter with him.

Such is his condition during the warm weather, but on the advent of cold or adverse climatic influences, bronchitis or broncho-pneumonia make their appearance, and the child shows the symptoms and physical signs of these diseases, and wasting takes place.

This condition has to be separated from chronic pleurisy with effusion; for, as regards auscultatory signs, there may be but little to choose between them; thus dulness of fluid character, impaired or nearly absent vesicular murmur, with a few bronchitic signs, may be the order of the day, or there may be distant tubular or hollow breathing. As far as these signs go, to make a definite decision would be an impossibility.

With a cirrhosed lung and a vesicular murmur, this is conducted from the healthy side. Occasionally one has an opportunity of observing the solution of such a phenomenon at an autopsy.

The position of the cardiac impulse, and the positive or negative results obtained from one or more exploratory punctures, should determine as to its nature. That such a puncture is not without danger in cirrhotic cases has been amply demonstrated by the case I have narrated. Further, it must be borne in mind that a dry tapping does not of necessity mean absence of fluid. The behavior of the needle after its passage through the chest-wall, whether into a cavity or solid lung, must be carefully noted. Further, the withdrawal of pus does not mean of necessity free pus in the pleural cavity; the pus may come from a dilated tube, or what not. The use of this instrument in an intelligent in contradistinction to a perfunctory manner should prove of the utmost assistance.

Further, this disease has to be separated from an invasion of new growth in the lung. Attention directed to the mediastinum, which is almost always attacked, should prevent any difficulty in this direction.

Sometimes ulceration of the dilated tubes takes place, and then, with offensive purulent sputa, a hectic temperature, and severe wasting, the child rapidly passes down hill. A microscopical examination of the sputa will in these cases reveal yellow elastic tissue. The sputa will not contain tubercle bacilli. Of course, however, it is quite possible, and highly probable in some cases, that tubercular infection has taken place secondarily, though the possibility of tuberculosis being the *fons et origo mali* must be well borne in mind. If such were the case, it is highly probable, though of course not absolutely certain, that the fellow lung in advanced disease would commence to show signs of implication.

From pneumonia of delayed resolution to fibroid pneumonia there is, as I have shown, a wide gap; and yet, not so wide after all, in some instances. Much will depend upon individual cases, and it is by no means an easy question to foretell the probable, or perhaps rather the possible, course of any consolidation.

That a consolidation can remain in abeyance for very many months, and then finally clear to physical signs, is a fact of very great importance indeed, and should at least teach the lesson of not too hastily jumping to a conclusion. On the other hand, in some cases, a few weeks alone have been found necessary to bring about any decided fibrosis of the organ. The pneumonia of delayed resolution is, *per se*, not dangerous; it becomes so if resolution does not come to its assistance. In such cases, as far as my own experience goes, the rubbing in of Scott's ointment, or some other mercurial ointment, has appeared to me a remedy of very considerable value. It has roused the dormant activity of the parts and has been attended by results of an encouraging nature. At this stage it is obvious that the line of treatment must be directed to the induction of such a desirable termination. There is no doubt that the longer the powers of resolution are deficient, the greater is the danger of a smouldering, though terribly destructive process going on. In addition, the general health must be improved in every way by tonics; iodide of potassium internally, good feeding, and change of air. For the latter, Newquay, in Cornwall, is an ideal resort. I do not confine patients to their beds under such circumstances. Full expansion of the lungs should be invited by gentle exercise, short of fatigue and of exposure to east winds and to damp and wet.

With a fibroid lung, all remedies must of necessity be palliative rather than curative. But much can be done even here, by placing the patient under suitable hygienic conditions. Thus cold, damp, and wet are to be avoided absolutely. Residence in a warm and equable climate, such as Ilfracombe, Torquay, the south of France, or Egypt, will at least make life endurable, and prevent the advent of bronchitic or broncho-pneumonic attacks, which are terribly frequent and harassing under adverse circumstances.

Tonics, suitable clothing, and good food, must be chiefly relied upon. With fair general health there is much less liability to bronchitic attacks. Acute attacks must be dealt with on the usual lines. With bronchorrhœa, inhalations of carbolic acid, creasote, iodine, and turpentine may be used with advantage, and the latter may be administered internally. Excessive coughing must be alleviated by opiates and chloral, and indications of cardiac failure combated by the usual remedies.